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ABSTRACT. The relationship of passive smoking to respiratory conditions and pulmonary function was assessed using a cross-sectional design in the defined population of Tecumseh, Michigan. The study population was made up of 3,482 children who were 0 to 19 yr of age at the 1962-1965 examination and for whom questionnaire information was available for both parents. Nearly 62% of children in this age group were exposed at the time of examination to at least 1 parent who smoked. Passive exposure to cigarette smoke was associated with an elevated prevalence of phlegm, wheeze, asthma, and chest colds among males and wheeze, bronchitis, and chest colds among females. Using logistic regression, offspring were shown to be 1.5 to 2.0 times more likely to have a respiratory condition if both their parents currently smoked than if both parents never smoked. FEV1 and FVC among males and Vmax50 among females were significantly lower by 5% in nonsmokers 10 to 19 yr of age whose parents were current smokers compared with similar offspring of never smoking parents. Respiratory conditions were generally more frequent and the level of lung function was generally lower for males from households where only mothers smoked compared with males from households where only fathers smoked, although sample size was limited. In females similar relationships were less consistent. Differences tended to be larger and more often significant for males than for females when respiratory symptoms and illness were examined. Comparisons between offspring of 2 current and 2 never smoking parents and those involving the number of parental smokers in a child's lifetime provided stronger associations of passive smoking with respiratory conditions and lung function than did the number of household smokers, duration, or amount of parental smoking. In general, these associations were independent of parental education, family size, parental reporting bias, and the child's own smoking habits.

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Passive Smoking in Childhood

Respiratory Conditions and Pulmonary Function in Tecumseh, Michigan[1-4]

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SUMMARY The relationship of passive smoking to respiratory conditions and pulmonary function was assessed using a cross-sectional design in the defined population of Tecumseh, Michigan. The study population was made up of 3,482 children who were 0 to 19 yr of age at the 1962–1965 examination and for whom questionnaire information was available for both parents. Nearly 62% of children in this age group were exposed at the time of examination to at least 1 parent who smoked. Passive exposure to cigarette smoke was associated with an elevated prevalence of phlegm, wheeze, asthma, and chest colds among males and wheeze, bronchitis, and chest colds among females. Using logistic regression, offspring were shown to be 1.5 to 2.0 times more likely to have a respiratory condition if both their parents currently smoked than if both parents never smoked. FEV, and FVC among males and $\dot{V}max_{75}$ among females were significantly lower by 5% in nonsmokers 10 to 19 yr of age whose parents were current smokers compared with similar offspring of never smoking parents. Respiratory conditions were generally more frequent and the level of lung function was generally lower for males from households where only mothers smoked compared with males from households where only fathers smoked, although sample size was limited. In females similar relationships were less consistent. Differences tended to be larger and more often significant for males than for females when respiratory symptoms and illnesses were examined. Comparisons between offspring of 2 current and 2 never smoking parents and those involving the number of parental smokers in a child's lifetime provided stronger associations of passive smoking with respiratory conditions and lung function than did the number of household smokers, duration, or amount of parental smoking. In general, these associations were independent of parental education, family size, parental reporting bias, and the child's own smoking habits.

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Introduction

Until recently, relatively few studies have focused on the health effects of passive or involuntary smoking. Passive smoking during infancy and childhood has been associated with acute respiratory illness (1–16), chronic respiratory symptoms (17–22), and reduced pulmonary function (21, 23–26), although not all investigations have confirmed these associations (27–31). The qualitative and quantitative differences of mainstream and sidestream cigarette smoke have been documented (32, 33); a number of constituents are more concentrated in sidestream than in mainstream smoke. The need for better characterization of exposure and control of potential confounding factors has been recognized (34–37).

Investigations involving young children are of interest for several reasons: (*1*) confounding effects of active smoking and occupational exposures are absent, (*2*) children may be more exposed and/or susceptible than adults, and (*3*) the risks of passive exposure can be assessed during the period of lung growth and development. Children spend 60 to 80% of their time indoors (38), depending on season and geographic location. Because cigarette smoking is prevalent among adults, the likelihood of passive exposure in children is high. It has been estimated that 54 to 70% of children are exposed to one or more cigarette smokers in the household environment (1, 27, 28, 38, 39). Because of the large number of exposed persons, the proportion of time spent indoors and recent energy conservation efforts, the public health impact of passive smoking could be substantial.

The purpose of this study was to assess the cross-sectional relationships of passive smoking to respiratory symptoms, illnesses, and lung function in children and adolescents of Tecumseh, Michigan. Several measures of passive smoking were developed from questionnaire data. Effects of parental education, family size, parental symptoms and illnesses, and active smoking by the children themselves are evaluated.

Methods

Study Population

Residents of Tecumseh, Michigan have been participants in a community-based prospective investigation for the past 25 yr. The major purpose of the Tecumseh Community Health Study has been to identify determinants of health and disease in a natural community. Its design, methods, and historical perspective have been described previously (40–42). Standard questionnaires, certain physiologic measurements, and clinical assessments by physicians were available.

During the second cycle of examinations, conducted between 1962 and 1965, a total of 4,378 children and adolescents 0 to 19 yr of age were interviewed and examined. Of the 4,378 subjects, the following were excluded from this investigation: 82 because they were not residing with their parents, 688 because both parents were not interviewed, and 126 because they were active smokers (smoking habits were available only for those 16 to 19 yr of age; children 15 yr of age or younger were assumed to be nonsmokers). A total of 3,482 nonsmoking males and females 0 to 19

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[3] Presented in part at the Annual Meeting of the American Thoracic Society, Kansas City, Missouri, May 1983.

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yr of age and members of their households constituted the study population.

Questionnaire

Personal, demographic, and medical information were ascertained using a standard questionnaire. Parents responded for children 15 yr of age or younger. Several respiratory symptoms and illnesses were selected for evaluation: these included cough, phlegm, wheeze, asthma, bronchitis, and colds settling in the chest. In general, questions involved a past history of these conditions, rather than ascertainment of symptoms and illnesses defined only at the time of interview. Specific questions used to define these respiratory conditions and results concerning neonatal, allergic, and other conditions are reported elsewhere (43). The diagnostic criteria for asthma, reported by Higgins and Keller (44) and Broder and coworkers (45), were used, and a probable or suspect diagnosis was included as asthma.

Information was available concerning parental education, family size, and presence or absence of parental respiratory symptoms or illness. Categories of parental education included: (*1*) at least one parent who did not complete high school, (*2*) both parents completed high school, and (*3*) either parent attended college. Family size was defined as the number of persons residing in a household. Categorical definitions were used to classify children according to whether their mothers or fathers had a history or diagnosis of the specific respiratory condition under study.

Pulmonary Function

A Wedge® spirometer (Med-Science Electronics, Burlington, MA) and a two-channel recorder (Sanborn Co., Waltham, MA) operating at a paper speed of 25 mm/s were used to measure lung function. Following maximal inspiration, subjects performed several maximal expiratory efforts until 2 satisfactory tracings were obtained. Measurements of volume and flow were based on the tracing with the largest vital capacity and were adjusted to body temperature and pressure saturated with water vapor (BTPS).

Seven volume and flow measurements were available (46); forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and forced expiratory flow at 50% of vital capacity ($\dot{V}max_{50}$) were selected for use in this study because they have been used in other studies and were available at subsequent examinations. Lung function analyses reported here involved young persons 10 to 19 yr of age in 1962 through 1965.

Measures of Passive Smoking

Children were classified by smoking habits of their household members at the time of interview. Only cigarette smoking and not pipe or cigar smoking was taken into account. Five measures of passive smoking were developed.

I. Current and Past Parental Smoking Habits:

Father:	Never	Current	Current	Never	All
Mother:	Never	Current	Never	Current	Others

II. Number of Parental Smokers During Child's Lifetime: (0, 1, and 2).

III. Number of Current Household Smokers: (0, 1, 2, 3, or more).

IV. Duration of Parental Smoking During Child's Lifetime.

V. Current Amount of Parental Smoking.

The first index provided one of the more extreme contrasts in passive smoke exposure, where children having both parents who never smoked are compared with those having both parents who were current smokers. Children from households where only fathers smoked and where only mothers smoked could also be compared using this index. The "all others" category included children having one or both parents who were former smokers. For the second index, children were categorized by presence or absence of parental smoking during the child's lifetime. To include potential prenatal exposure to parental smoking, a child's lifetime was defined as 1 yr before birth to the time of examination in 1962–1965. Smoking habits of siblings and relatives who were 16 yr of age or older were included with those of parents in the classification of current household smokers. For duration and amount of parental smoking, the number of years and average number of cigarettes smoked per day by each parent were summed.

Data Analysis

Prevalence rates of respiratory symptoms and illnesses, and levels of lung function in children and adolescents were compared across parental smoking categories using 5-yr, age- and sex-specific groups. Stratification was used initially to control for potential confounding by parental education, family size, parental history of respiratory symptoms or illness, and active smoking by adolescents. Significance was assessed using standard *t* and chi-square tests for differences between means and proportions, respectively. Age-adjusted prevalence rates were derived using the age distribution of all nonsmoking subjects 0 to 19 yr of age examined in 1962–1965 as a standard. A Cochran/Mantel-Haenszel procedure was used to test for the average partial association between passive smoking and a specific respiratory symptom or illness, controlling for the effects of age group and assessing whether a linear trend exists (47). To compare age-adjusted means, variances for those having or not having a specific respiratory condition were calculated, and a standard *z* statistic for comparing means with known but unequal variances was used to determine significance.

Multiple logistic regression (48) was employed to control for potential confounders simultaneously. Sex-specific analyses were performed using each respiratory condition as the response variable. The number of parental smokers during a child's lifetime (0, 1, 2) was selected as the independent variable of primary interest and was coded using 2 indicator variables. Parental education, family size, parental symptom or illness, as well as age of the child were considered as covariates. For asthma a diagnosis of hay fever and history of other allergies were also included in the model as potential predictors. Several methods of including age were considered; regression coefficients were similar for each. Likelihood ratio tests (48) were used to compare methods of including age, to decide whether certain variables should be retained in the logistic model, and to confirm whether or not statistical interaction was present. Goodness of fit for these logistic models was assessed using methods proposed by Lemeshow and Hosmer (49), where information from both cases and noncases is incorporated.

Levels of lung function were expressed as a percent of predicted or were adjusted using analysis of covariance. Predicted values of FEV_1, FVC, and $\dot{V}max_{50}$ were obtained by regressing observed values of asymptomatic nonsmokers on age and height separately for 2 age and sex groups using the population of nonsmokers 10 to 19 yr of age who were

TABLE 1

REGRESSION STATISTICS FOR LUNG FUNCTION MEASUREMENTS, ASYMPTOMATIC NONSMOKING MALES AND FEMALES, TECUMSEH, 1962–1965*

Sex	Age (yr)	Measure	Regression Statistics					
			n	a	b_1	b_2	R^2	$S_{y \cdot x}$
Male	10–15	FEV_1	528	−5.293	0.115	0.043	0.747	0.423
		FVC	528	−6.447	0.118	0.052	0.739	0.507
		$\dot{V}max_{50}$	516	−4.311	0.152	0.038	0.490	0.729
	16–19	FEV_1	127	−3.722	—	0.046	0.289	0.534
		FVC	127	−5.214	—	0.064	0.303	0.720
		$\dot{V}max_{50}$	124	−1.226	—	0.034	0.075	0.890
Female	10–15	FEV_1	524	−3.688	0.093	0.032	0.550	0.396
		FVC	524	−4.322	0.096	0.038	0.559	0.437
		$\dot{V}max_{50}$	514	−2.816	0.112	0.031	0.264	0.754
	16–19	FEV_1	155	−3.397	—	0.040	0.233	0.432
		FVC	155	−3.305	—	0.041	0.220	0.459
		$\dot{V}max_{50}$	154	−2.134	—	0.037	0.054	0.940

* Regression model: Predicted lung function = $a + b_1 \cdot$ age (yr) $+ b_2$ (height in cm) for 10- to 15-yr-olds; predicted lung function = $a + b_1 \cdot$ height (cm) for 16- to 19-yr-olds.

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TABLE 2

PREVALENCE OF PARENTAL SMOKING AMONG SUBJECTS 0 TO 19 YR OF AGE, TECUMSEH, 1962–1965

Parental Smoking			
Father	Mother	n	%
Never	Never	567	15.7
Current	Current	1,136	31.5
Current	Never	983	27.2
Never	Current	109	3.0
All others		813	22.5
Total		3,608	99.9

free of asthma and wheeze without colds, and if 16 to 19 yr of age, free of cough, phlegm, and moderate or severe shortness of breath. A total of 1,357 nonsmokers 10 to 19 yr of age met these criteria and had complete age, height, and lung function data (FEV_1, FVC, or $\dot{V}max_{50}$). The significant terms of the selected regression model included age and height for those 10 to 15 yr of age and height only for those 16 to 19 yr of age. Regression statistics for these models are presented in table 1. Models that included powers of height, weight, or interaction terms did not increase substantially the amount of variation explained by the simple model employing only age and height. Race was not included in the models because all subjects are white. Regression statistics for FEV_1 and $\dot{V}max_{50}$ were published previously for nearly the same group of children 10 to 15 yr of age (50). Sex-specific regressions were used because of differences between males and females in lung growth.

Analysis of covariance models were also used to adjust levels of lung function for age and height, parental education, and family size (51). The covariance model assumes no interaction and a linear relationship between covariates and lung function. Tests for equality of slopes among those exposed and unexposed to parental smoking were performed to rule out interaction; linearity was also assessed. Both assumptions for the model were met (43). In contrast to the percent of predicted method of adjustment, this approach does not require definition of a healthy standard population and allows comparisons to be made in units of actual lung volume or flow.

Results

Prevalence of Passive Smoking

Prevalence of passive exposure to cigarette smoke was estimated using two-parent households where both parents were interviewed (table 2). A total of 61.7% of all subjects 0 to 19 yr of age had at least one currently smoking parent; 31.5% had both parents who currently smoked. Having a father as the only parental smoker was far more prevalent than having a mother as the only parental smoker (27.2% versus 3.0%). Only 15.7% of the subjects 0 to 19 yr of age were never exposed to parental smoking.

Respiratory Symptom and Illness Prevalence

Prevalence rates of several respiratory symptoms and illnesses in Tecumseh have been shown previously to vary with age and sex (43). Age-specific prevalence rates of several respiratory conditions are presented in table 3. For most respiratory conditions, prevalence rates tended to be higher in males than in females, significantly so for phlegm, wheeze, asthma, and chest colds in at least one of the age groups. Cross-sectional frequencies of phlegm, wheeze, and chest colds tend to decrease as age increases, whereas with cough, asthma and bronchitis prevalence rates vary with age in a less consistent manner. Age-adjusted prevalence rates of 4 respiratory conditions are presented by parental smoking category for males and females in figures 1 and 2, respectively. For both sexes and all 4 conditions, prevalence rates were higher among nonsmoking children whose parents both currently smoked than among children whose parents never smoked. Differences

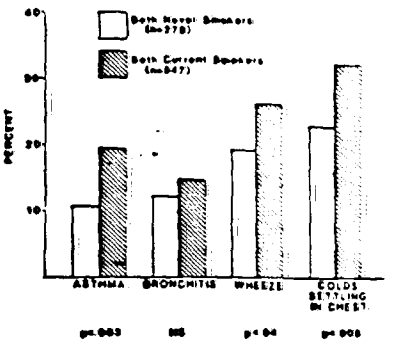

Fig. 1. Age-adjusted prevalence of respiratory conditions by parental smoking, male nonsmokers 0 to 19 yr of age, Tecumseh, 1962–1965.

were significant for the majority of conditions.

When the number of parental smokers during a child's lifetime was considered, age-adjusted prevalence rates were highest for children exposed to 2 parental smokers and generally lowest for unexposed children, the trend being significant for phlegm, wheeze, asthma, and colds settling in the chest among males and for wheeze, bronchitis, and colds settling in the chest among females (table 4). Although not presented in detail here, for all respiratory conditions except bronchitis in males, parents of nonsmokers 0 to 19 yr of age reported smoking significantly more cigarettes (mean differences ranged from 1 to 4 per day) and for significantly longer periods of time (mean differences ranged from 7 to 24 months) when a given respiratory symptom or illness was reported for their children than when it was not reported, after adjusting for differences in age ($p < 0.0001$).

Previous work in Tecumseh has shown that parental smoking habits are related to parental education but not to family size (43, 52). In this investigation, level of education was highest among households where both parents never smoked. Although not shown here, children from households where both parents currently smoked tended to have higher respiratory symptom and illness prevalence rates than those where both parents never smoked within each degree of parental education and family size.

When results were stratified by parental history of a given respiratory condition, there was some reduction in the magnitude of the parental smoking effect, yet for several conditions the relationship remained significant. For example, in households where both parents

TABLE 3

PREVALENCE (%) OF RESPIRATORY SYMPTOMS AND ILLNESSES BY AGE AND SEX, NONSMOKING CHILDREN FROM TWO-PARENT HOUSEHOLDS, TECUMSEH, 1962–1965*

	0–4		5–9		10–14		15–19	
Respiratory Condition	M (n = 470)	F (n = 491)	M (n = 555)	F (n = 540)	M (n = 482)	F (n = 460)	M (n = 241)	F (n = 243)
Cough	7.6	8.6	10.7	9.5	10.8	7.8	7.3	7.8
Phlegm	18.4	13.5†	14.1	14.9	13.7	9.7	8.5	8.9
Wheeze	31.7	24.9†	20.6	18.5	19.5	15.0	17.9	16.2
Asthma	13.6	10.7	16.2	9.2‡	17.0	9.7‡	13.3	8.2
Bronchitis	13.3	10.5	15.9	12.6	11.0	10.0	11.7	14.5
Chest cold	39.9	30.4‡	29.4	26.5	20.9	19.0	19.2	14.9

* Differences tested between males and females within each age group using chi-square
† $p < 0.05$
‡ $p < 0.01$
§ $p < 0.001$

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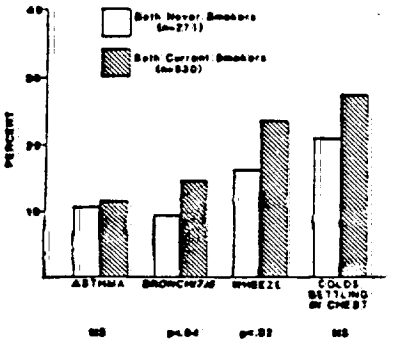

Fig. 2. Age-adjusted prevalence of respiratory conditions by parental smoking, female nonsmokers 0 to 19 yr of age, Tecumseh, 1962–1965.

reported a history of phlegm, male offspring had a prevalence rate for phlegm of 13.4, 12.5, and 18.5% for 0, 1, and 2 parental smokers, whereas the prevalence rate was 14.9, 9.0, and 17.5%, respectively, when both parents denied history of phlegm. Differences remained significant for phlegm and asthma among males regardless of a history of the same symptom or illness in their parents.

Results obtained using logistic regression models are presented in table 5. The odds ratios represent measures of the degree of association between passive smoking and each respiratory condition controlling for potential confounding by age, parental education, and family size. For example, the odds of a male 0 to 19 yr of age having asthma are 2.16 times as great if he was passively exposed to 2 parents who currently smoked than if he was unexposed. For both sexes and almost all respiratory conditions, odds ratios were higher for children with 2 parental smokers compared with children who had never been exposed to parental cigarette smoke. Odds ratios tended to be higher for males from households where mothers were the only smokers than for males from households where only fathers smoked. The pattern was reversed, though less consistently, for females.

When logistic regression models employing the number of parental smokers during a child's lifetime (0, 1, or 2) were used as a measure of passive smoking, similar odds ratios were obtained for most respiratory conditions. When children with one parental smoker were compared with the unexposed reference group, odds ratios were frequently close to or less than 1.0, yet did not differ significantly from 1.0. This suggests that exposure to one parental smoker, who was most often the father, is not associated with an increased probability of having these respiratory symptoms or illnesses. In comparing logistic models using the 2 different passive smoking measures, the −2 log likelihood values and the fraction of variance explained by the models are almost identical, suggesting that little is gained statistically by categorizing parental smoking more completely with 5 as opposed to 3 levels (43). An analysis of the goodness of fit for these logistic models revealed close agreement between observed and expected cases across deciles of risk.

Pulmonary Function

Mean lung function expressed as a percent of predicted is presented in figure 3 for nonsmoking males and females 10 to 19 yr of age whose parents were both never (98 males and 93 females) or current (201 males and 199 females) smokers. Mean FEV_1 and FVC for males and $\dot{V}max_{50}$ for females were significantly lower if both parents were current smokers rather than never smokers. Results were virtually identical when 10- to 14- and 15- to 19-yr-old age groups were analyzed separately.

Levels of FEV_1 and FVC for males and $\dot{V}max_{50}$ for females were inversely related to the number of parental smokers during a child's lifetime among nonsmokers 10 to 19 yr of age. Using analysis of covariance to adjust levels of lung function for age and height, male nonsmokers 10 to 19 yr of age from households where both parents smoked had a mean FEV_1 that was 144 ml (4.6%) lower than that for males with no parental smokers (table 6). Similarly, a deficit of 173 ml (4.9%) in

TABLE 4

AGE-ADJUSTED PREVALENCE (%) OF RESPIRATORY CONDITIONS BY SEX AND NUMBER OF PARENTAL SMOKERS DURING CHILD'S LIFETIME, NONSMOKERS 0 TO 19 YR OF AGE, TECUMSEH, 1962–1965*

	Males			Females		
	Number of Parental Smokers			Number of Parental Smokers		
Respiratory Condition	0 (n = 339)	1 (n = 718)	2 (n = 681)	0 (n = 360)	1 (n = 716)	2 (n = 648)
Cough	8.6	8.4	11.4	8.4	8.0	8.7
Phlegm	13.4	12.8	18.8†	8.7	13.5	13.6
Wheeze	20.9	20.4	26.5†	16.0	17.3	22.9‡
Asthma	13.4	11.3	20.9§	9.8	7.9	11.7
Bronchitis	12.0	11.9	15.2	8.8	11.3	13.2†
Chest cold	23.4	27.6	32.0‡	20.0	22.4	27.4‡

* Generalized Cochran/Mantel-Haenszel test for average association in three-way contingency tables
† $p < 0.05$
‡ $p < 0.01$
§ $p < 0.001$.

TABLE 5

ODDS RATIOS RELATING PARENTAL SMOKING TO RESPIRATORY CONDITIONS ADJUSTING FOR THIRD VARIABLES USING MULTIPLE LOGISTIC REGRESSION, NONSMOKING MALES AND FEMALES 0 TO 19 YR OF AGE, TECUMSEH, 1962–1965*†

		Parental Smoking				
Sex	Respiratory Condition	Father: Never Mother: Never	Current Current	Current Never	Never Current	All Others
Male	Cough	1.0	1.38	0.89	0.66	1.20
	Phlegm	1.0	1.62‡	0.93	1.77	1.15
	Wheeze	1.0	1.47‡	1.04	1.42	1.21
	Asthma	1.0	2.16‡	0.84	1.37	1.19
	Bronchitis	1.0	1.23	0.88	1.46	1.03
	Chest cold	1.0	1.56‡	1.16	1.30	1.37
Female	Cough	1.0	1.17	1.06	0.95	1.30
	Phlegm	1.0	1.43	1.92‡	0.81	1.44
	Wheeze	1.0	1.60‡	1.01	1.41	1.10
	Asthma	1.0	1.05	0.65	1.16	0.79
	Bronchitis	1.0	1.75‡	1.61	0.73	0.74
	Chest cold	1.0	1.39	1.19	0.90	1.00

* Odds ratios are relative to the reference category where both parents were never smokers.
† Third variables include age, parental education, and family size for all except asthma where age, hay fever, and other allergies were used.
‡ Significant at $p < 0.05$.

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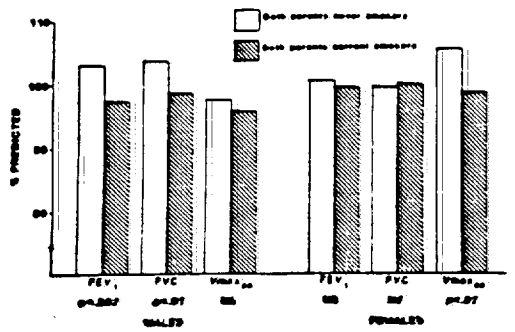

Fig. 3. Mean % predicted lung function by parental smoking, male and female nonsmokers 10 to 19 yr of age, Tecumseh, 1962–1965.

FVC for males 10 to 19 yr of age and of 185 ml/s (5.1%) in $\dot{V}max_{50}$ for females 10 to 19 yr of age was observed.

Although not presented here, mean level of lung function tended to be inversely related to the total number of smokers in the household; this was most evident for FEV_1% predicted among male nonsmokers, yet sample size was small, and trends were not significant (43). Lung function was also inversely related to duration and amount of parental smoking among nonsmoking males 10 to 19 yr of age but not among females (43).

Differences in lung function across parental smoking categories were similar in magnitude when results were stratified across levels of parental education and family size. When each potential confounder was included in an analysis of covariance, model associations between passive smoking and impaired lung function persisted.

Discussion

Prevalence rates of respiratory conditions and level of lung function have been examined in a defined community for nearly 3,500 young persons in relation to the smoking habits of their parents. The prevalence of passive exposure to cigarette smoke in Tecumseh during the 1962–1965 time period was similar to that found in other population surveys (1, 27, 39). Approximately 62% of those 0 to 19 yr of age in this study had at least one parent who currently smoked compared with 54 to 70% reported in other studies. Given the large proportion of children exposed, the amount of time spent indoors, especially by younger age groups, and recent energy conservation efforts, which reduce ventilation, the public health impact of passive smoking could be substantial.

Several indirect measures of passive exposure to household cigarette smoke were developed from questionnaire data. Most investigations to date have used either a dichotomous classification or the number of current parental smokers as exposure variables; only one study defined exposure to parental smoking with reference to the child's lifetime (25). A few studies have classified exposure based on the number of cigarettes currently smoked per day (5, 9, 18, 53). Although results of this investigation were generally similar for all measures of passive smoking, current and past smoking habits and the number of parental smokers during a child's lifetime were most useful in assessing passive smoking and respiratory outcomes. Misclassification of exposure was a potential problem both for this investigation and others preceding it. The frequency of contact between children and their parents while cigarette smoking occurred, as well as exposure patterns in day care settings for young children of working parents, are additional factors that should be addressed in future research.

Passive exposure to cigarette smoke was associated with increased prevalence of phlegm, wheeze, asthma, and colds settling in the chest among males, and wheeze, bronchitis, and colds settling in the chest among females in Tecumseh. Several cross-sectional studies have demonstrated significant associations between parental smoking and phlegm (17), wheeze (17, 21), bronchitis (5–7), and asthma (12, 15), whereas others have not documented such associations (16, 27). The lack of significant association between parental smoking and history of cough in this study was consistent with results of several studies (16, 27) but not with those of others (17–19, 22). Cameron and Robertson (4) were among the first to suggest that differences in illness prevalence related to parental smoking might be of greater magnitude in geographic locations where more time is spent indoors because of the climate.

The significant inverse relationship observed in this investigation between parental smoking and level of lung function in nonsmokers 10 to 19 yr of age is consistent with several previous studies (21, 23–26, 54) but not with others (17, 27, 28, 30). Most of the studies showing a positive association also demonstrated a dose-response relationship. Results of

TABLE 6

MEAN (± SE) LUNG FUNCTION IN CHILDREN ADJUSTED FOR AGE AND/OR HEIGHT USING ANALYSIS OF COVARIANCE BY NUMBER OF PARENTAL SMOKERS, TECUMSEH, 1962–1965*

Sex	Age (yr)	Parental Smokers (n)	Examined (n)	FEV_1 (L)	FVC (L)	$\dot{V}max_{50}$ (L/s)
Male	10–14	0	70	2.812 ± 0.047	2.924 ± 0.055	3.105 ± 0.083
		1	197	2.582 ± 0.028	2.675 ± 0.033	3.227 ± 0.049
		2	180	2.480 ± 0.030	2.790 ± 0.034†	3.060 ± 0.057
	15–19	0	41	4.210 ± 0.084	4.841 ± 0.108	4.866 ± 0.141
		1	106	4.057 ± 0.052	4.843 ± 0.067	4.611 ± 0.088
		2	75	4.052 ± 0.062	4.828 ± 0.080	4.499 ± 0.105
	10–19	0	111	3.139 ± 0.043	3.565 ± 0.053	3.626 ± 0.074
		1	303	3.080 ± 0.026	3.466 ± 0.032	3.686 ± 0.045
		2	255	2.995 ± 0.028‡	3.392 ± 0.035‡	3.537 ± 0.049
Female	10–14	0	85	2.373 ± 0.048	2.579 ± 0.053	3.365 ± 0.094
		1	181	2.380 ± 0.028	2.583 ± 0.032	3.238 ± 0.056
		2	169	2.368 ± 0.029	2.581 ± 0.033	3.187 ± 0.058
	15–19	0	50	3.041 ± 0.057	3.267 ± 0.062	4.074 ± 0.127
		1	109	3.003 ± 0.039	3.246 ± 0.042	3.852 ± 0.086
		2	72	3.039 ± 0.048	3.285 ± 0.051	3.853 ± 0.106
	10–19	0	115	2.807 ± 0.037	2.818 ± 0.040	3.614 ± 0.076
		1	290	2.802 ± 0.023	2.826 ± 0.025	3.457 ± 0.048
		2	241	2.809 ± 0.025	2.835 ± 0.028	3.429 ± 0.052†

* Defined during child's lifetime excluding smokers 16 to 19 yr of age
† $p < 0.05$
‡ $p < 0.01$

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this research tend to support these findings for several measures of lung function, although the minimal level at which an effect of parental smoking can be detected remains unclear. Investigators have suggested that the lack of association observed in several studies may be due to climatic factors (17, 27), selection of a pulmonary function measure (peak expiratory flow), which may not be adequately sensitive (30), or small sample size (55). Schilling and coworkers (28) did not show a significant relationship in general, although significantly lower maximal flow at 50% of FVC (MEF_{50} or $Vmax_{50}$) was observed among nonsmoking girls whose mothers smoked. Tager and coworkers (55) suggested that a larger sample might have revealed a similar relationship among boys.

Measures of lung function in Tecumseh children were not entirely independent, because values for children of the same household were correlated. Intraclass correlation coefficients of approximately 0.25 have been observed between siblings, with 0.46 between male siblings and 0.66 between female siblings in Tecumseh (44). When a random sample of one child per household was selected from this population, results were essentially unchanged, demonstrating that a lack of independence would not account for the observed association with passive smoking.

Several investigators have adjusted children's lung function by their parent's lung function (28) or by their parent's body mass (56). It is likely, as suggested by Weiss and associates (57), that this adjustment would mask a true passive smoking effect, should it exist.

Several investigators have attempted to control for potential confounders of the relationship between passive smoking and respiratory outcomes. For example, Fergusson and colleagues (5) demonstrated an increase in bronchitis and/or pneumonia during the first 2 yr of life in children of mothers who smoked after controlling for socioeconomic status, family size, and maternal age. Several investigators have shown that the association of passive smoking with impaired lung function remains significant after controlling for parental education (23, 24), sibship size (21, 23), and the child's own smoking (25). **Results from this study indicate that associations of parental smoking with prevalence of respiratory conditions and lung function are independent of parental education and family size.**

Cigarette smoking by children may be related to both parental smoking and the respiratory outcomes under study. In this research, significant relationships between measures of passive smoking and respiratory outcomes have been demonstrated among children and adolescents who were presumed to be never smokers. There is a possibility that some of the observed passive smoking effect in children 10 to 15 yr of age might be due to unreported active smoking, and some of the effect in those 16 to 19 yr of age might be due to inaccurate reporting of their smoking habits. In subsequent analyses, subjects who were reexamined 15 yr later and reported cigarette smoking at an age that was younger than their baseline examination in 1962–1965, were also excluded and, in general, results were unchanged. It is unlikely that observed associations between respiratory conditions and parental smoking among the youngest age groups could be explained by active smoking by the children themselves.

Parents who smoke and report respiratory symptoms or illnesses themselves may tend to overreport respiratory conditions in their children; this parental reporting bias has been raised as a possible explanation for passive smoking health effects (6, 7, 16, 17, 27, 57). Schenker and associates (16) suggested that associations between passive smoking and respiratory symptoms and illnesses may be due to shared genetic and/or environmental factors, or to overreporting by symptomatic parents for their children. Weiss and colleagues (58) found an increased risk of atopy in nonsmoking children of smoking mothers that was not explained by maternal reporting bias. When stratification or logistic regression was used in the present research to control for parental reporting bias, trends were occasionally diminished, yet relationships generally remained significant.

It is possible that some of the observed association with passive smoking might be due to gas cooking or heating. The relationship between gas cooking or heating and respiratory conditions (16, 29, 53, 59–61), and associations with lung function have been demonstrated in some studies (23, 29) but not in others (54, 55, 59, 62). Results of a pilot study involving a sample of 213 nonsmoking women from Tecumseh did not show a significant relationship between gas cooking and FEV_1 (62). Schenker and associates (16) did not find gas cooking to be an independent risk factor for chronic respiratory symptoms or illnesses.

In general, respiratory conditions were more prevalent and, although not reported here, lung function was lower for male offspring when the only smoker in a household was the mother rather than the father (43). Stronger associations with maternal smoking than with paternal smoking have been observed in a number of studies (5, 15, 16, 21, 23, 53–55, 61, 63, 64). This is consistent with a potentially greater passive exposure of children if their mothers smoke than if their fathers smoke, because of more time spent by offspring in the presence of their mothers during this time period. In this study, associations of passive smoking with respiratory conditions and lung function appeared stronger among younger than among older age groups and among males rather than among females. Higher prevalence rates of respiratory symptoms and illnesses have been observed among younger age groups (2, 5, 21) and among male children (21, 65). Younger age and male sex have been identified as independent risk factors for acute respiratory illnesses and chronic respiratory symptoms (16). Associations between maternal smoking and lung function were strongest among younger male children in one study (64) and among female children in others (21, 24, 26, 54).

The apparent sex difference observed in this research was not explained by a difference in dose or duration of exposure to parental smoking. Confounding by active smoking could account for some of the significant associations observed in males 10 to 15 yr of age if a greater proportion of males than of females were active smokers. However, such confounding would not account for such associations in the younger age groups. As suggested by Taussig and coworkers (66), the sex differential in response to parental smoke exposure may have a physiologic basis. The higher prevalence and incidence of asthma among males, for example, may be consistent with an increased susceptibility of males to sidestream cigarette smoke or other pollutants. Weiss and associates (58) recently demonstrated significantly elevated odds ratios for atopy in males but not in females with a prior history of bronchiolitis or croup.

The magnitude of association between parental smoking and children's lung function appears similar to that found in other studies. Whether a decrement of 144 ml in FEV_1 (4.6%) associated with having 2, compared with no, parental smokers will become clinically significant with increasing age remains to be inves-

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tigated. Tager and associates (55) recently reported a deficit in expected growth of lung function in children participating in the Six-Cities Study. Additional longitudinal studies should be conducted to further substantiate these long-term adverse effects and to quantitate the magnitude of impact that exposure to parental cigarette smoke may have on respiratory health. More accurate estimates of passive smoking using specifically designed questionnaires and biochemical markers such as cotinine in urine or saliva are needed.

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